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APPLICATION NO.	FILING DATE	FIRST NAMED INVENTOR	ATTORNEY DOCKET NO.	CONFIRMATION NO.
10/525,278	11/21/2005	Stephan Schwers	71325-015	7785

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EXAMINER	
BAUSCH, SARAE L	
ART UNIT	**PAPER NUMBER**
1634	

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Please find below and/or attached an Office communication concerning this application or proceeding.

The time period for reply, if any, is set in the attached communication.

Office Action Summary	Application No.	Applicant(s)	
	10/525,278	SCHWERS ET AL.	
	Examiner	Art Unit	
	SARAE BAUSCH	1634	

-- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --

Period for Reply

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE *3* MONTH(S) OR THIRTY (30) DAYS, WHICHEVER IS LONGER, FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133). Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

Status

1)☒ Responsive to communication(s) filed on *28 April 2008*.

2a)☐ This action is **FINAL**. 2b)☒ This action is non-final.

3)☐ Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

Disposition of Claims

4)☒ Claim(s) *17-110* is/are pending in the application.

4a) Of the above claim(s) *21-84 and 86-110* is/are withdrawn from consideration.

5)☐ Claim(s) _____ is/are allowed.

6)☒ Claim(s) *17-20 and 85* is/are rejected.

7)☐ Claim(s) _____ is/are objected to.

8)☐ Claim(s) _____ are subject to restriction and/or election requirement.

Application Papers

9)☒ The specification is objected to by the Examiner.

10)☐ The drawing(s) filed on _____ is/are: a)☐ accepted or b)☐ objected to by the Examiner.

Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).

Replacement drawing sheet(s) including the correction is required if the drawing(s) is objected to. See 37 CFR 1.121(d).

11)☐ The oath or declaration is objected to by the Examiner. Note the attached Office Action or form PTO-152.

Priority under 35 U.S.C. § 119

12)☒ Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).

a)☒ All b)☐ Some * c)☐ None of:

1.☒ Certified copies of the priority documents have been received.

2.☐ Certified copies of the priority documents have been received in Application No. _____.

3.☐ Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).

* See the attached detailed Office action for a list of the certified copies not received.

Attachment(s)

1) ☒ Notice of References Cited (PTO-892)
2) ☐ Notice of Draftsperson's Patent Drawing Review (PTO-948)
3) ☐ Information Disclosure Statement(s) (PTO/SB/08) Paper No(s)/Mail Date _____.
4) ☐ Interview Summary (PTO-413) Paper No(s)/Mail Date. _____ .
5) ☐ Notice of Informal Patent Application
6) ☐ Other: _____.

DETAILED ACTION

1. This action is in response to applicants correspondence mailed 04/28/2008.

Election/Restrictions

2. Applicant's election of group I in the reply filed on 04/28/2008 is acknowledged. Because applicant did not distinctly and specifically point out the supposed errors in the restriction requirement, the election has been treated as an election without traverse (MPEP § 818.03(a)).

3. Applicant's election with traverse of SEQ ID No. 469-42 (baySNP11594) in the reply filed on 04/28/2008 is acknowledged. The traversal is on the ground(s) that they are entitled to a reasonable number of species and that the restriction requirement should be an election of species. This is not found persuasive because each of the baySNPs recited in the claims amplify nucleic acid sequences that have a different structure and do no share a common structure. Additionally, each of the nucleic acids that are amplified by the primers recited in the claims do not share a common property or activity, thus each nucleic acid is a distinct invention owing to the functional and structural differences between the nucleic acids .

The requirement is still deemed proper and is therefore made FINAL.

4. Claims 21-84 and 86-110 are withdrawn from further consideration pursuant to 37 CFR 1.142(b), as being drawn to a nonelected invention, there being no allowable generic or linking claim. Applicant timely traversed the restriction (election) requirement in the reply filed on 04/28/2008.

5. Currently, claims 17-20 and 85 are under examination. Claim 85 is under examination with respect to SEQ ID No. 469-472, baySNP11594 (see section 8 below).

Specification

6. This application does not contain an abstract of the disclosure as required by 37 CFR 1.72(b). An abstract on a separate sheet is required.

Claim Objections

7. Claim 85 objected to because of the following informalities: claim 85 recites SEQ ID No. 469-42 (baySNP11594) however it appears as though SEQ ID No. 469-42 is a typo and the claim should recite SEQ ID No. 469-72. Appropriate correction is required.

8. It is noted that the specification teaches baySNP11594 is amplified using SEQ ID No. 469-472 not SEQ ID No. 469-42 as recited in the claims (see table 2a paragraph 633 amendment mailed 11/21/2005). Thus the claim has been interpreted to require the primers for amplification of baySNP11594 requiring SEQ ID No. 469-472 not 469-42. Thus, instant claim 85 is being examined as using the oligonucleotide primers SEQ ID No. 469-472 baySNP11594.

Claim Rejections - 35 USC § 112- Enablement

9. The following is a quotation of the first paragraph of 35 U.S.C. 112:

> The specification shall contain a written description of the invention, and of the manner and process of making and using it, in such full, clear, concise, and exact terms as to enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to make and use the same and shall set forth the best mode contemplated by the inventor of carrying out his invention.

10. Claims 17-20 and 85 are rejected under 35 U.S.C. 112, first paragraph, as failing to comply with the enablement requirement. The claim(s) contains subject matter which was not described in the specification in such a way as to enable one skilled in the art to which it pertains, or with which it is most nearly connected, to make and/or use the invention.

Factors to be considered in determining whether a disclosure meets the enablement requirement of 35 USC 112, first paragraph, have been described by the court in *In re Wands*, 8 USPQ2d 1400 (CA FC 1988). *Wands* states at page 1404,

> "Factors to be considered in determining whether a disclosure would require undue experimentation have been summarized by the board in Ex parte Forman. They include (1) the quantity of experimentation necessary, (2) the amount of direction or guidance presented, (3) the presence or absence of working examples, (4) the nature of the invention, (5) the state of the prior art, (6) the relative skill of those in the art, (7) the predictability or unpredictability of the art, and (8) the breadth of the claims."

The nature of the invention and the breadth of the claims

The claims are drawn to a method of calculating a patient's relative risk for adverse drug reactions from statin therapy by genotyping a SNP in DNA of the patient. The claims are further drawn to a SNP that is a C or T and genotypes 1, 2, and 3, wherein 1 is CC, 2 is TT and 3 is CT. The claims are further limited to genotyping using the primers SEQ ID No. 469-472, baySNP11594.

The rejected claims encompass analysis of any patient, including human and non- human. The rejected claims encompass analysis of any statin therapy and any adverse drug reaction. The rejected claims encompass analysis of any genotype in any gene.

The nature of the claims requires the knowledge of a correlation between detection of the presence of any SNP and adverse drug reaction with statin therapy.

The invention is in a class of inventions which the CAFC has characterized as "the unpredictably arts such as chemistry and biology" (Mycolgen Plant Sci., Inc. v. Monsanto Co., 243 F.3d 1316, 1330 (Federal Circuit 2001)).

Guidance in the Specification

The specification teaches that the aim of pharmacogenomics is to produce personalized medicines whereby administration of the drug class and dosage is tailored to an individual genotype (see paragraph 24, USPgPub US2007/0128597 A1, it is noted the paragraph numbering in this rejection will refer to the US2007/0128597 A1 paragraphs). The specification teaches that statins reduce the primary and secondary risk of coronary artery disease and coronary events (see paragraph 26) and teaches that the tolerability of these drugs during long term administration is an important issue, adverse reactions involving skeletal muscle are not uncommon and myopathy as well as rhabdomyolysis may occur as well as increase in serum creatine kinase, athralgia, and elevation of liver transaminase has been associated with statin administration (see paragraphs 26-28). The specification asserts that the present invention provides diagnostic tests to predict a patient's individual response to statin therapy and responses include but not limited to adverse drug reaction (see paragraph 30). However the specification does not teach that any genotype in any gene is associated with an adverse drug reaction from statin therapy. The specification does provide guidance on how any genotype in any patient, human or non-human, is predictably correlated statin efficacy response in order for the skilled

artisan to conclude a an adverse drug reaction to administration of statin based on the presence of the relative risk of a genotype.

The specification asserts that candidate genes were analyzed and discovery of polymorphisms that lie in genomic regions of unknown physiological function. The specification asserts that after conducting an association study of polymorphic sites in a number of candidate genes showed a strong correlation with the phenotypes healthy, cardiovascular disease prone, high responder to statin, low responder to administration of statin, tolerant patient, and adverse drug reaction patient (see paragraph 43). The specification teaches that ADR patient has diagnosis of muscle cramp, muscle pain, muscle weakness, myalgia, myopahty or serum CK levels hither than 140 mg/dl in women and 160 mg/dl in men (see table 1b). The specification teaches that baySNP numbers refer to an internal numbering of the phenotype SNPs and different polymorphisms are found in the association study, specifically baySNP 11594 is adverse drug reaction SNP with genotype CC, CT, or TT of the phosphomevalonate kinase gene (see table 3). However, the specification does not provide any guidance on how to predictably correlate any genotype with relative risk of developing adverse drug reaction in any gene, nor predictably correlate any SNP within the phosphomevalonate kinase gene(PVMK). The specification does not describe the position or numbering of the SNP associated with baySNP 11594 nor what location within the gene the CC, CT, or TT genotype is found. The specification does not provide any guidance on the type of statin that was administered to the patients. The specification does not provide any guidance on determining a genotype in any patient other than human of baySNP 11594. Additionally, as will be discussed below, it is unpredictable to correlate any polymorphism with an adverse drug response.

The specification teaches the results of association studies in which subjects were homozygous or heterozygous for a particular SNP in reference to baySNP 11594 in the PVMK gene. However the specification does not teach the type of statin administered to the patients not teach the location of the baySNP 11594 in the PVMK gene for genotypes CC, CT, and TT. The specification does not teach a representative number of statins and detection of genotypes in all gene, much less using the baySNP 115924. While the specification demonstrates a study of adverse drug reaction of baySNP 11594 (see table 5a, 5b), it does not however teach a method of determining genotype CC, TT, or CT of baySNP 11594 as the specification does not teach the location of the genotype of baySNP 11594 within the gene PVMK much less teach any location of any SNP in any gene in any patient to determine adverse drug reaction in ay patient. It is unclear based on examples and data presented in the tables, as well as throughout the specification, the location of the SNPs within the gene of interest and which genotype infers a statin response because it is not clear which position within the gene the tables and data refer to, much less the type of statin administered. The specification does not provide guidance of which genotypes within the baySNPs are analyzed with regard to the data presented and it is unclear what statin determines adverse drug reaction. Given the lack of clear distinction with regard to the data presented in the specification and the lack of guidance in the specification with regard to determining which genotype is analyzed in each gene, it is unclear which genotype is considered to be predictive of any statin response.

Working Examples

The specification demonstrates a working examples of genotyping several subjects with adverse reaction to administration of statin. However, the examples in the specification do not

clearly recite which sequence, location of SNP, or statin is being examined. The tables merely recite genotype of a specific gene, for example baySNP 11594 but the specification is silent in teaching the location of the SNP within the gene. For example, table 6a shows baySNP11594 relative risk for diagnostic conclusion of particular patient. The specification asserts that baySNP 11594 has no risk (see table 6a, 6b). Thus, the specification demonstrates that it is unpredictable to association CC or CT or TT with adverse drug reaction in any patient in any gene, particularly PVMK, baySNP 11594. The specification provides no guidance on which genotype is associated with a relative risk of adverse drug reaction in any statin as reciting baySNP is not descriptive, and it is unclear which genotype this refers to – is one of the CC at position 101? 140? 160? what position is this? which sequence is this? The specification does not explain the tables or the data associated within the tables

The unpredictability of the art and the state of the prior art

The prior art teaches that there are many parameters that need to be evaluated prior to using a genetic test to determine a disease and that these parameters yield gaps in information that are needed to complete a thorough screening of a genetic test. Post filing art, Thompson et al. (The Pharmacogenomics Journal, 2005, pp. 1-7) teach that variation in individual response to statin therapy has been widely studied for a potential genetic component (see abstract). Thompson et al. teach that associations of genotype with statin response results summarized from 22 studies covering more than 20 genes revealed that only one gene, apoE was identified in more than one study and nearly as many studies with apoE were negative as were positive. Thompson et al. teach that the largest study of 1536 patients for 148 SNPs across 10 genes found that only two highly linked SNPs in the HMGCR gene were associated with pravastatin response

(see 1st column, 1st full paragraph, pg. 2). Thompson et al. teach despite the multitude of papers reporting significant associations between statin response and genotype, very few replication studies have been published. Thompson et al. teach that analysis of replication rates reveals that only a small fraction of published associations were replicated and the issues that plague association studies have been well described, such as chance, bias, and confounding effects (see pg. 6, 1st column, last paragraph cont'd to 2nd column, 1st paragraph). The SNPs tested by Thompson et al. were attempts to replicate previous findings and the only statin response association that has been noted repeatedly in the literature is SNPs rs7412 and rs429358 in apoE. Thompson et al. teach that differences in details of population, length of treatment, statin doses, and size of groups examined will vary the results of a genetic study (see pg. 6, 2nd column, 2nd full paragraph). Thompson et al. further teaches that while pharmcogenomics has been very successful at predicting efficacy or safety for some drugs like warfarin, thiopurines, and codeine, none of the association found here predict atorvastatin LDL-C lowering in a manner sufficient to impact decisions on treatment. Thompson et al. teach that age and gender are equally good at predicting response and these effects could be due to compliance with drug regimen rather than intrinsic differences among those groups (see pg. 6, 2nd column, 3rd full paragraph). Based on the data presented in the specification and the prior art teachings, it is unpredictable to correlate the presence of a haplotype to infer any statin response, as the specification does not teach a large sample size with family-based studies, along with statistical methods or confidence levels greater than 95%. Furthermore, the post-filing art study conducted by Thompson et al. teaches that to date there is only two SNPs that repeatedly are replicated, rs7412 and rs429358 which suggest that SNPs are not predictable in inferring a statin response since the results can not be replicated.

Furthermore, Ionnidis (Plost Med, 2005, 2(8):e124) teach that most published research findings are false. Ionnidis et al. teach that ill-founded strategy of claiming conclusive research finding solely on the basis of a single study assed by formal statistical significance represented and summarized by p values (see pg. 0696, 2nd column, 1st full para.) Ionnidis et al. teach that research findings are likely to be true that in fields that undertake large studies, such as randomized controlled trials (several thousand subjects randomized) than in small studies such as sample sizes 100 fold or smaller (see pg. 0697, 3rd column, 2nd full para.) Ionnidis et al. teaches that what matters is the totality of evidence and that statistical significance of a single study only gives a partial picture (see pg. 0701, 1st column). Additionally, Hattersley et al. (Lancet, 2005, vol 366, pp. 1315-1323) teaches that the key quality in an association study is sample size (see page 1318, 2nd column, 1st full paragraph). Hattersley et al. teach that sample sizes of thousands are needed to detect variants that are common but have low relative risk and teach that allelic odds ratio of 1.1 to 2.0 requires the number of controls to be in thousands (see page 1318, 2nd column, 1st full paragraph and table 3). Hattersley et al. teach that apparent studies in identifying interesting associations with studies much smaller than implied by table 3 (in the thousands) might suggest that calculations are too pessimistic and small initial studies rarely find the correct result and even when they do they are likely to overestimate the true effect size (see page 1318, 1st column, 1st full paragraph). Hattersley et al. further teaches that emphasis has been on the need for greater stringency in the association studies in order to prove a given association and suggest a p value of $5x10^{-8}$, however arguments from Bayesian perspective suggest that $5x10^{-5}$ should be sufficient to constrain the false discovery rate.

In the instant case, the specification does not provide a study that includes a large sample size, predictable p values or familial studies, as recommended by the art to determine an association between any genotype and statin response.

Neither the art nor the specification teach that any genotype or any genotype of the phosphomelvonate kinase is predictably correlated to determine patient's relative risk of adverse drug reaction from statin response.

Level of Skill in the Art

The level of skill in the art is deemed to be high.

Quantity of Experimentation

Given the lack of guidance in the specification with regard to correlating the presence of a genotype to determining statin response along with the evidence in the art with regard to predictably correlating genetic assays with associations, the quantity of experimentation in this area is extremely large. The skilled artisan would have to perform an extremely large study and include different populations to determine if in fact there was either an association between any genotype in any gene in any patient with an adverse statin response. The results of such a study are clearly unpredictable as evidence by the post filing art (which reflects the current state of the art) and the teachings in the specification with regard to lack of sample size, statistical analysis, or correlation of statin response to presence of gene variations. Post filing art, Thompson et al. teaches that to date there is only two SNPs correlated with statin response that repeatedly replicated in the literature, rs7412 and rs429358. Thompson et al. further teaches that while pharmcogenomics has been very successful at predicting efficacy or safety for some drugs like warfarin, thiopurines, and codeine, none of the association found here predict atorvastatin LDL-

C lowering in a manner sufficient to impact decisions on treatment. Thompson et al. teach that age and gender are equally good at predicting response and these effects could be due to compliance with drug regimen rather than intrinsic differences among those groups (see pg. 6, 2nd column, 3rd full paragraph). In the instant case, it would be unpredictable as to whether or not the any genotype, particularly HUMPMK1 presented in the specification would be responsible for identifying an adverse statin response. The skilled artisan would have to take into account gender, familial, and ethnic studies, as evidence by Thompson et al. and the instant specification does not teach a correlation any genotype with statin response. Thompson et al. teach that differences in details of population, length of treatment, statin doses, and size of groups examined will vary the results of a genetic study (see pg. 6, 2nd column, 2nd full paragraph). The skilled artisan would then have to screen multiple SNPs, to determine those that is associated between the presence of a SNP and statin response. Given the lack of guidance in the specification and the post filing art with respect to accurately testing genetic diseases, such analysis is replete with unpredictable experimentation and is considered undue.

Claim Rejections - 35 USC § 102

11. The following is a quotation of the appropriate paragraphs of 35 U.S.C. 102 that form the basis for the rejections under this section made in this Office action:

> A person shall be entitled to a patent unless –
>
> (b) the invention was patented or described in a printed publication in this or a foreign country or in public use or on sale in this country, more than one year prior to the date of application for patent in the United States.

12. Claims 17-20 are rejected under 35 U.S.C. 102(b) as being anticipated by Walter (Eur. Heart Journal, 2001, vol. 22, pp. 587-595).

Walter et al. teach a method of genotyping the P1A2 polymorphism which is a C for T at position 1565 in exon 2 of the glycoprotein IIIa gene (see pg. 589, genotyping of the GP IIb/IIIa polymorphism) (genotyping a SNP in a DNA from a patient) (claim 18- 20, wherein the SNP is C to T and CC, TT, and CT). Walter et al. teach that 142 of the patient carry the P1A2 allele and 7 patients were homozygous for P1A2 allele (see baseline characteristics) (genotyping three possible genotypes for each SNP). Walter et al. teach statistical analysis of the data including risk factors (see data and statistical analysis, pg. 589). Walter et al. teach genotyping for P1A polymorphism is useful tool to target statin therapy in high risk patients. Walter et al. teach P1A2 allele have a beneficial effect of statins, thus have a relative risk of less than 1 (see pg. 594, 1st column, 2nd paragraph and tables 3-5).

13. Claims 17-20 are rejected under 35 U.S.C. 102(b) as being anticipated by Pedro-Botet et al. (Atherosclerosis 2001 vol. 158:183-193).

Pedro-Botet teaches a method of genotyping genomic DNA from human patient to analyze for the presence of ApoE E2, E2, and E4 (see 2.2, pg. 185) (three genotypes comprising a CC, TT, and CT, claims 18-20). Pedro-Botet et al. teach statistical analysis of ApoE E2, E3, and E4 genotype to test treatment and allele main effected and allele by treatment interactions (see section 2.3, pg. 185) (determining relative risk associated with each genotype). Pedro-Botot et al. teach those with E3 and E4 are less responsive to atorvastatin (see pg. 192) (adverse drug reaction).

Double Patenting

14. A rejection based on double patenting of the "same invention" type finds its support in the language of 35 U.S.C. 101 which states that "whoever invents or discovers any new and useful process ... may obtain a patent therefor ..." (Emphasis added). Thus, the term "same invention," in this context, means an invention drawn to identical subject matter. See *Miller v. Eagle Mfg. Co.*, 151 U.S. 186 (1894); *In re Ockert*, 245 F.2d 467, 114 USPQ 330 (CCPA 1957); and *In re Vogel*, 422 F.2d 438, 164 USPQ 619 (CCPA 1970).

A statutory type (35 U.S.C. 101) double patenting rejection can be overcome by canceling or amending the conflicting claims so they are no longer coextensive in scope. The filing of a terminal disclaimer cannot overcome a double patenting rejection based upon 35 U.S.C. 101.

15. Claims 17-20 provisionally rejected under 35 U.S.C. 101 as claiming the same invention as that of claim 17-20 of copending Application No. 11/572039. This is a provisional double patenting rejection since the conflicting claims have not in fact been patented. Claims 17-20 of '039 are identical in scope to instant claims 17-20.

16. Claims 17-20 provisionally rejected under 35 U.S.C. 101 as claiming the same invention as that of claim 17-20 of copending Application No. 10/524302. This is a provisional double patenting rejection since the conflicting claims have not in fact been patented. Claims 17-20 of '302 are identical in scope to instant claims 17-20.

Conclusion

17. No claims are allowable.

Any inquiry concerning this communication or earlier communications from the examiner should be directed to Sarae Bausch whose telephone number is (571) 272-2912. The examiner can normally be reached on M-F 9am-5pm.

If attempts to reach the examiner by telephone are unsuccessful, the examiner's supervisor, Ram Shukla can be reached on (571) 272-0735. The fax phone number for the organization where this application or proceeding is assigned is (571) 273-8300.

Information regarding the status of an application may be obtained from the Patent Application Information Retrieval (PAIR) system. Status information for published applications may be obtained from either Private PAIR or Public PAIR. Status information for unpublished applications is available through Private PAIR only. For more information about the PAIR system, see http://pair-direct.uspto.gov. Should you have questions on access to the Private PAIR system, contact the Electronic Business Center (EBC) at (866) 217-9197 (toll-free).

Any inquiry of a general nature or relating to the status of this application or proceeding should be directed to (571) 272-0547.

Patent applicants with problems or questions regarding electronic images that can be viewed in the Patent Application Information Retrieval system (PAIR) can now contact the USPTO's Patent Electronic Business Center (Patent EBC) for assistance. Representatives are available to answer your questions daily from 6 am to midnight (EST). The toll free number is (866) 217-9197. When calling please have your application serial or patent number, the type of document you are having an image problem with, the number of pages and the specific nature of the problem. The Patent Electronic Business Center will notify applicants of the resolution of the problem within 5-7 business days. Applicants can also check PAIR to confirm that the problem has been corrected. The USPTO's Patent Electronic Business Center is a complete service center supporting all patent business on the Internet. The USPTO's PAIR system provides Internet-based access to patent application status and history information. It also enables applicants to view the scanned images of their own application file folder(s) as well as general patent information available to the public.

For all other customer support, please call the USPTO Call Center (UCC) at 800-786-9199.

/Sarae Bausch/
Primary Examiner, Art Unit 1634